systolic (58 (19) ml) volumes and LV mass (134 (42) g). CK (median (range)) at 8-12 hours (620 (74-1344) IU/l) and at the MR scan (228 (57-2508) IU/l) correlated with LE volume: r=0.78, p=0.0001 and r=0.70, p=0.002, respectively. CK (111 (69-4869) IU/l) on admission was unrelated to LE volume. CKMB (median (range)) at 8-12 hours (54 (3.4-226) mg/ml) and at the MR scan (11 (1.2-406) mg/ml) correlated with LE volume: r=0.59, p=0.01 and r=0.58, p=0.02, respectively. CKMB on admission was unrelated to LE volume.

Conclusions. Plasma concentrations of TnI measured 8-12 hours after onset of chest pain are very closely related to the volume of late enhancement measured by MR suggesting that the currently recommended timing of TnI sampling does provide an accurate reflection of infarct size.

1170-101 Homozygote Mutation of C161→T Polymorphism in the Exon 6 of Peroxisome Proliferator-Activated Receptor γ3 Gene Is Associated With Onset of Premature Myocardial Infarction

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Background: Peroxisome proliferator-activated receptor (PPAR) is a nuclear receptor. Activation of PPAR γ with ligands could modulate some gene transcription, thereby leading to multiple antiatherogenic and fibrinolytic effects. However, the association between effect of C161→T mutation of the exon 6 of PPARγ3 and the onset of premature myocardial infarction is not clarified.

Methods: We recruited 146 patients (pts) (mean age 45 yr, 129 male) with premature myocardial infarction (MI) (onset age ≤ 50 yr) and 146 control subjects (mean age 45.3 yr, 120 male). Polymerase chain reaction and restriction fragment length polymorphism were used to define C161→T polymorphism.

Results: The frequency of the PPAR γ3 TT genotype among pts with premature MI was significantly higher than that in control subjects (13% vs 5.5%, odds ratio [OR] 2.6, 95% confidence interval [CI] 1.1 to 6.1, p=0.03). This association was not observed in CC, or CT genotypes. The homozygote TT genotype (OR 3.1, 95% CI 1.2 to 7.9), smoking (OR 3.5, 95% CI 2.1 to 6.0), hypertension (OR 3.6, 95% CI 1.9 to 6.9), and diabetes mellitus (OR 3.5, 95% CI 1.5 to 8.4) were independent risk factors for premature MI after adjustment of conventional risk factors.

Conclusion: There was a significant association between the PPAR γ3 C161→T homozygote mutation and the onset of premature MI in our population. Effect of the TT genotype on the production and activity of PPAR γ warrants further investigation.

1170-102 Reduced Post-Infarction Myocardial Apoptosis in Women: A Clue to Their Different Clinical Course?

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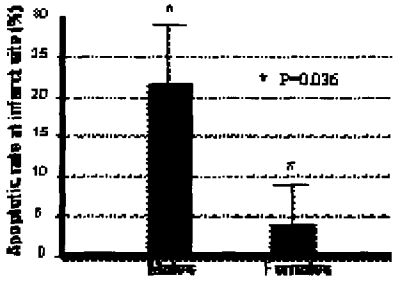
Background. Ischemic heart failure (HF) is less common and severe in women. Myocardial apoptosis has a fundamental role in post-infarction remodeling, however, little is known about gender-modulation of post-infarction apoptosis. We investigated whether apoptosis late after acute myocardial infarction (AMI) is different in females in comparison to males.

Methods. Hearts from 21 men and 9 women dying 10 days-2 months after AMI were examined at autopsy. Apoptotic rate (AR) at infarct site and apparently normal myocardium was assessed with *in-situ* end labeling for DNA fragmentation (TUNEL) and co-staining for caspase-3. We also performed co-stainings for PCNA and SC-35 to identify potential false positive results at TUNEL. **Results.** AR in infarcted areas was 5-fold higher in males than in females (21.2% vs 4.3%,p=0.036). See Figure. This difference remained statistically significant even at multivariate analysis (p=0.020) and was particularly evident in subjects with persistent infarct-related artery (IRA) occlusion.



Conclusions. Myocardiocyte apoptosis after AMI is more severe in men in comparison to women. This difference may explain the more aggressive and progressive course of post-infarction HF in men and the relatively more benign LV remodeling in women.

1170-103 Relation of Growth Hormone and Insulin-Like Growth Factor-1 to Ventricular Remodeling in Acute Myocardial Infarction

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Background Experimental data have suggested that early treatment with growth hormone (GH) and insulin-like growth factor-1(IGF-1) could attenuate ventricular remodeling after acute myocardial infarction. Our aim was to determine whether serum GH and IGF-1 level are related to ventricular remodeling in patients with acute myocardial infarction.

Methods: Within 24 hours of chest pain onset, 52 consecutive patients were studied at baseline, on day 5, and at 6 weeks after the acute myocardial infarction. GH and IGF-1 were measured and related to positive ventricular remodeling defined as an increase ≥5% in left ventricular diastolic volume (LVDV), left ventricular systolic volume (LVSV), their variations (Δ EDV and Δ ESV) and ejection fraction (EF) evaluated by echocardiography (Sympson's method) on day 3 and 6 weeks.

Results: Median value of GH was higher at baseline (2.19 ng/ml) and significantly decreased at day 5 (0.26 ng/ml) and 6 weeks (0.55 ng/ml) (p<0.001). IGF-1 significantly increased from baseline: mean 136±90 ng/ml, to day 5: mean164±95 ng/ml and 6 weeks: mean 200±89 ng/ml, p< 0.0001). Patients with baseline IGF-1 below the median value showed less EF at 6 weeks than patients above the median: 46±11% versus 53±9.8% respectively (p=0.01), and significant increase ESV: ΔESV 9±18 versus ΔESV 2±15 (p=0.02). At day 5, IGF-1 below the median value was associated with a significant increase ESV: ΔESV 11.35±14 versus ΔESV 0.11±18 (p=0.04).

Patients with positive ventricular remodeling showed significant lower levels of IGF-1(117±84ng/ml) than those with negative remodeling (165±93ng/ml) (p=0.05) at baseline, and day 5 (152±111ng/ml versus 183±61ng/ml respectively) (p=0.03). No significant differences were found at 6 weeks. No association was found between GH levels, ventricular remodeling and ejection fraction at baseline, day 5 and 6 weeks.

Conclusions: There was a strong association of low serum levels of IGF-1 with left ventricular remodeling and impaired ventricular function at baseline and day 5. No relation was found with GH, a labile and unstable biological marker. This results could suggest a protective role of IGF-1 to prevent the early remodeling process after myocardial infarction.

1170-104 ST Elevation Myocardial Infarction Caused by Spontaneous Distal Embolization From Unstable Lesions: Frequency and Sequelae of a Distinct Pathophysiological Entity

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Background: Distal embolization is an important feature in acute myocardial infarction. Previously, we demonstrated that distal embolization after primary angioplasty resulted in larger infarct size. However, distal embolization may in part be due to angioplasty. Therefore, we investigated the characteristics of patients with distal embolization before primary angioplasty.

Methods: We assessed angiographic data of 631 patients. Distal embolization before angioplasty was defined as a distal filling defect with an abrupt "cut-off" in at least one of the peripheral coronary branches of the infarct related artery, distal to the infarct related segment. Follow-up was obtained at 30 days.

Results: Distal embolization before angioplasty was visible in 30 (5%) patients. They were younger than patients without distal embolization (56y vs. 61y, p=0.04). Patients with distal embolization before angioplasty had a higher rate of TIMI 2 or 3 flow before angioplasty (57% vs. 31%, p=0.005), more often thrombus (53% vs.12%, p1 (20% vs. 7%, p<0.03) and had a higher 30 day mortality rate (10% vs. 2%, p=0.03).

Conclusions: Distal embolization before angioplasty can be visualized in 5% of the patients with acute myocardial infarction. It is associated with more thrombus, a lower angioplasty success rate and heralds a higher rate of intracoronary stenting. It is associated with more Killip class>1 and higher 30 days mortality. Patients with distal embolization before angioplasty are a distinct subset of patients with a high rate of complicated myocardial infarction.

1170-105 Marked Reduction in No-Reflow With Late Initiation of Hypothermia in a Rabbit Myocardial Infarct Model

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Background: Hypothermia in conjunction with reperfusion (rep) is a novel approach for treatment of acute myocardial infarction now being tried in humans. Whether reflow, an important predictor of clinical outcome, is changed when hypothermia is initiated late during ischemia is unknown. This study tests if myocardial hypothermia, starting near the end of ischemia and continuing for 2 hr of rep, protects microvasculature reducing no-reflow. Methods: Rabbit hearts received 30 min coronary artery occlusion/ 3 h rep. Regional hypothermia (H, n=10) started 10 min before rep and continued for 2 hr of rep was compared with normothermia (N, n=10). Regional myocardial blood flow (microspheres) was measured during occlusion and at the end of rep. The anatomic zone of no-reflow (thioflavin S in vivo injection) and zone of macroscopic hemorrhage were measured in the LV. Results: Myocardial temperature (° C) in H was decreased by 5.0±0.4 from baseline (36.9±0.2) and remained about 32° during the cooling phase, returning to 36.3±0.3 at 3 hr. N hearts remained within 0.2° of baseline (37.4±0.1) throughout. Both groups were equally ischemic during occlusion, but at the end of rep reflow to the previously ischemic zone was significantly higher in H, 69±6% of normal blood flow vs 34±5% in N (p=.0001). The zone of anatomic no-reflow was significantly smaller in H, 12±4% of the ischemic zone vs 37±3% in N (p=.0001), and the macroscopic zone of hemorrhage